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CARBOHYDRATE METABOLISM IN SUBMARINER PERSONNEL

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INTRODUCTION

Submariners have been shown to have an increased rate of glucose utilization 2 hours after an oral loading test⁽¹⁾. Relative hypoglycemia due to excess insulin production has been considered a first indication of developing diabetes⁽²⁾. However, diabetes can not be defined in terms of blood glucose level alone but rather in its relationship to insulin release, utilization, and effectiveness^(2,3). The ability to interpret insulin response to a glucose load by separating normal from abnormal patterns provides for disease detection at its earliest identifiable point when maximum benefit might result from appropriate management⁽⁴⁾.

→ The present study was undertaken to reevaluate potential pre-diabetes in submariners and assess the extent of defects in their glucose metabolism. ←

MATERIALS AND METHODS

Fifty-eight active duty submariners between the ages of 22 and 46 who had made no fewer than five FFM patrols and fifty-eight comparable non-submariners were studied. Of the submariners 35 were on a sea duty tour.

Following an overnight fast blood samples were taken. The subjects then consumed 100 gm. glucose. Blood was again drawn 1 and 2 hours after ingestion of the sugar.

Body weight, height, and abdominal circumference were measured. A questionnaire answered tendencies to gain or lose weight, and exercise habits. The subjects were categorized as having none or little, moderate, heavy, or daily heavy and lengthy exercise.

Glycosylated hemoglobin was measured in the fasting blood samples by column chromatography while glucose and insulin were determined enzymatically and by RIA, respectively, in the fasted and post-prandial serum.

Percent body fat was calculated using body weight and abdominal circumference⁽⁵⁾. Each subject was categorized as obese or normal

weight; Obesity being defined as 115% desirable body weight. The mean of the range for "medium frame" from the Tables of Desirable Weights for Men and Women (Metropolitan Life Insurance Co.) was considered 100%.

RESULTS

Despite significant differences in body weights, no differences were found between the submariners and non-submariners with respect to age, percent body fat, degree of fatness, glycosylated hemoglobin, or family history of diabetes. Highly significant differences in the amount of exercise were found between the two groups.

Significantly higher serum glucose levels occurred one and two hours following the glucose challenge in the submariners as compared to controls. Additionally, the submariners exhibited a delay in the return of insulin levels two hours after the glucose load. In an attempt to classify the nature of the defect in carbohydrate metabolism the Wilkerson Point System, for glucose values, used in conjunction with patterns of insulin response described by Kraft(4) serves as the means for classifying the type of defect. APPENDIX I

No significant differences between submariners and non-submariners occurred with respect to the amount of defect. However when the number of subjects with normal carbohydrate metabolism and the combined number of those with all carbohydrate defects are compared, 55% of the submariners exhibited a defect vs 45% of the controls. A Chi Square analysis reveals that 26% of the time could this ratio be attributed to chance alone. Generally, the more severe the defect the greater the percentage of submariners who exhibit it.

Regarding carbohydrate metabolism and fatness among the subject population as a whole, 35% normal weight and 61% obese exhibited defect ($p < .005$). A positive correlation was also found between carbohydrate defects vs. % body fat for both non-submariners (.520 $p < .001$) and submariners (.249 $p < .05$).

An inverse relationship between amount of exercise and carbohydrate metabolism characteristics occurred in both submariners and non-submariners.

An inverse relationship also seems to exist between exercise and severity of defect in both normal weight and obese.

DISCUSSION

At no time were any of the group mean values outside the limits of clinical normality and only three subjects of the entire population accumulated as much as one Wilkerson Point. At the same time a decrease in sensitivity to insulin in the submariners as a whole as seen by the higher one-and two-hour post-prandial glucose with increased two-hour insulin indicates some abnormality in these individuals as a group.

The effect of small population size may have contributed to the lack of statistical significance between the submariners and controls using Kraft's classification although a trend toward potential disease did occur in the submariners.

Differences between normal weight and obese individuals, for all subjects, with respect to defective carbohydrate metabolism reconfirms the positive correlations between obesity and diabetes have been frequently reported(6). The characterization of the subjects as normal weight or obese is used for comparative purposes(2,4). Although there was no difference between the two groups in fatness or % body fat, a somewhat more significant relationship exists between carbohydrate defects and % body fat in the non-submariners than in the submariners. This suggests that some factor in the submariner's environment or lifestyle such as fluctuating weight gain and loss may alter the relationship between amount of fat carried and the tendency to develop a carbohydrate defect.

The significantly greater amount of exercise by the non-submariners appears to play an important role in the maintenance of normal carbohydrate metabolism in these subjects. Exercise has a dramatic effect on glucose tolerance and increased insulin sensitivity in trained individuals(7). In the present study a significant negative correlation was also found between exercise vs one and two hour postprandial glucose and two hour insulin for all subjects in addition to the inverse relationship between exercise and carbohydrate defect. Despite reported insulin lowering effects of physical conditioning in obese subjects, their serum insulin concentration still remains higher than in comparably trained lean persons(7). That these individuals are not completely protected against defects in carbohydrate metabolism is further substantiated in our finding that at all exercise levels the obese exhibited a greater defect.

Conclusions: Submariners have a shift toward glucose intolerance resulting from increased peripheral insulin resistance and therefore a tendency toward development of carbohydrate metabolic defects. This increase in insulin resistance more likely results from decreased exercise among submariners rather than from increased carbohydrate intolerance associated with obesity since the submariners as a group were found to be no fatter than the controls.

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APPENDIX I

From: Kraft, J.R. Detection of diabetes mellitus in situ
 (occult diabetes).
 Laboratory Med. 6: 10-22; 1975

WILKERSON POINTS

TIME	PLASMA	POINTS
Fasting	130	1
1 hour	195	1/2
2 hours	140	1/2
3 hours	130	1/2

Two or more points is considered diagnostic of diabetes.

When abnormal insulin patterns are associated with normal glucose tolerances the results are considered indicative of prediabetes or occult diabetes (also called diabetes in situ), the earliest detectable phase of diabetes mellitus.

The criteria used are as follows:

I. NORMAL

WILKERSON PT	INSULIN PATTERN
0	Fasting level between 0-30 μ U Peak at either 1/2 or 1 hr. Sequential return to fasting levels at 2 to 3 hours. Two plus three hr value totals < 60 μ U

II. BORDERLINE

WILKERSON PT	INSULIN PATTERN
0 POINTS	Normal peak $2 + 3$ hr total > 60 μ U < 100 μ U

III. OCCULT OR PREDIABETES

WILKERSON PT	INSULIN PATTERN
0 POINTS	Normal insulin peak-delayed return $2 + 3$ hr total > 100 μ U
0 POINTS	2 hr delay in peak $2 + 3$ hr total > 100 μ U

0 POINTS	3 hr delay in peak 2 + 3 hr total > 100 μ U
0 POINTS	High fasting > 50 μ U 2 + 3 hr total > 100 μ U

IV. DIABETES

WILKERSON PT	INSULIN PATTERN
1/2-3 POINTS	Normal peak 2 + 3 hr total > 100 μ U
1/2-3 POINTS	2 hr peak delay 2 + 3 hr total > 100 μ U
1/2-3 POINTS	3 hr peak delay 2 + 3 hr total > 100 μ U
1/2-3 POINTS	High fasting > 50 μ U Peak delay 2 + 3 hr total > 100 μ U
2 or more POINTS	All values < 30 μ U 2 + 3 hr total < 60 μ U